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NEURAL CONTROL OF THE DIRECTION OF COVERT VISUAL  
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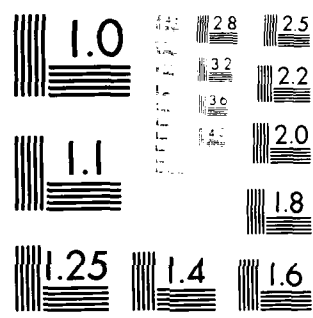
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Cognitive Science Program

NEURAL CONTROL OF THE DIRECTION  
OF COVERT VISUAL ORIENTING

Michael I. Posner

Technical Report 84-4

University of Oregon

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20. ABSTRACT (Continue on reverse side if necessary and identify by block number) In cases of unilateral parietal damage patients have difficulty in handling stimuli contralateral to the lesion. Our study shows a major problem is in disengaging attention from its current focus to deal with targets in a contralateral direction irrespective of the visual field in which the target occurs. This is true for both right and left-sided lesions. It is likely that the visual field and thus the hemisphere which first receives the target information is also important, but that is not clear in our results. The study confirms a suggestion by Kinsbourne (1977) that each		

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20. hemisphere directs attention in a contralateral direction. It implies that for directing attention the two hemispheres must be constantly interchanging control and thus sharing information from the two hemifields. These studies suggest the importance of control of the location of covert attention prior to the assessment of lateralization of cognitive functions.

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# Neural Control of the Direction of Covert Visual Orienting<sup>1</sup>

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Lesions of the parietal lobe have effects on the ability to attend to information that arises from locations in space contralateral to the lesion (see De Renzi, 1982 for a review). We have attempted to discover the specific nature of the attentional deficit involved (Posner, Cohen & Rafal, 1982; Posner, Walker, Friedrich & Rafal, 1983). Our studies have involved an experimental paradigm which has been used widely with normals (Posner, 1980) to study the ability to orient attention in visual space. It requires the person to fixate at a central location. Cues are introduced at different locations on the CRT display. The cues are thought to cause a shift of attention. The shift of attention is measured by examining the latency of response to target events that occur at the cued location in comparison to other locations at the same distance from fixation.

When patients with parietal lesions were studied using this paradigm we found a very great elevation in response time in cases when attention was drawn to positions in the visual field ipsilateral to the lesion and targets went to the contralateral field.

1. Draft of paper presented to Psychonomic Society, November 1983. This research supported by NIMH grant 1R01 MN38503-01 and ONR contract #N0014-83-K-1601.

Similar dramatic elevations in reaction time are also found when attention is cued to a location at fixation and targets are presented in the contralateral field as illustrated in Figure 1.

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 Insert Figure 1 about here  
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The effect of a central cue on a contralateral target rules out explanations which emphasize the power of the ipsilateral event to extinguish contralateral events, or are based on expectancy of the target location (since targets were equally probable on either side following a central cue) or eye movements in response to the ipsilateral cue.

We have considered the shift of visual attention produced by a peripheral cue to consist of three more elementary mental operations shown in Figure 2. These are disengagement from the current focus of attention, movement to the target location and engagement with the target.

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 Insert Figure 2 about here  
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Our finding that the main impairment in the cases of parietal lobe lesions occurs only when subjects are cued to an incorrect location suggests that the lesions main affect is on the ability of a target contralateral to the lesion to serve to disengage the person from the current attentional focus. Some of the patients also show a slowing of reaction times on the side contralateral to the lesion even after attention has been cued there. This suggests a deficit in the engagement function. However, other patients show no deficits in either the move or engage function,

but still show deficits in the disengagement operation.

These results suggest that the parietal lobe represents an important route by which attention can be oriented toward a visual stimulus. The current paper is addressed to additional details on how this is accomplished. One possibility is that stimuli coming directly to the lesioned hemisphere fail to reach attention in sufficient strength to produce a reorienting. This view is consonant with the term hemispheric inattention which is commonly applied to the syndrome resulting from parietal lesions (Weinstein & Freedland, 1977). A closely related theory suggests that what is important is not the hemisphere to which the stimulus is directly projected but the position of the stimulus with respect to the gravitational straight ahead or hemispace (Bowers, Heitman, & Van Den Abell, 1981). A third view suggests that the effect arises because each hemisphere controls the operations which orient covert attention in the contralateral direction. This view seems close to that suggested by Kinsbourne, 1977. Usually covert attention and overt attention are thought to be completely confounded since we tend to look at what we are interested in. However, the covert orienting paradigm discussed above allows a dissociation of the two and thus, a test of whether each hemisphere appears to control shifts of attention in a direction contralateral to the lesion (e.g., leftward for right side lesions).

In this experiment subjects look at a large cathode ray tube on which is plotted a central fixation cross flanked by three boxes located 3, 6 and 9 degrees to immediate left and right of



fixation. Each trial begins with 150 millisecond brightening of one of the six boxes. Either 100 or 600 millisecond following brightening a star is plotted in one of the boxes (target). The task is to respond to the target as quickly as possible. There are four general types of trials. On VALID trials the target appears at the cued location. On CROSS trials the cue occurs at the center position on one side and the target at the center position on the opposite side. On Move trials the cue occurs at the near or far position on one side and the target at the center position of the same side. The display, trial types and frequency of trial type are illustrated in Figure 3.

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 Insert Figure 3 about here  
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On two thirds of the trials in each block the time between cue and target was 100 millisecond and on the remaining one third of the trials it was 600 millisecond. The 100 millisecond interval insured that subjects could not shift their eyes between cue and target. The use of short and long SOA trials virtually eliminates anticipations at the short interval although a few occur for normals at the long interval.

The basic paradigm has been run on nine young normal subjects and on seven parietal patients who have been shown previously to have problems with disengaging attention to targets contralateral to the lesion.

The results for the nine normal subjects indicate that the only statistically significant effect other than SOA is the interaction between direction of movement and visual field

( $p < .05$ ). This interaction was replicated in another study of ten young normals. It indicates that targets involving movements outward from the near cue to center give systematically longer RTs than movements inward from the far cue to the center. Thus, normals show better orienting when the cue is further from the fovea than the target.

The results for the seven parietal patients are shown for valid trials (in comparison with normals) in Figure 4. The results for valid, cross and move trials at the two delay intervals are shown in Figure 5a, b. Statistical analysis shows main effects of interval ( $p < .01$ ), field ( $p < .01$ ) and condition ( $p < .001$ ).

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 Insert Figure 4 and 5a,b  
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At both intervals cross trials are longer than valid trials and this tends to be greater in the contralateral field than in the ipsilateral field. In general, times in the contralateral field are longer than in the ipsilateral. A sub analysis of the move trials shows RTs for movements in a direction contralateral to the lesion are slower than for those ipsilateral to the lesion ( $p < .05$ ). There is also an interaction between direction and field ( $p < .05$ ). Movements in the ipsilateral direction do not show any difference between the two fields. This effect is very striking when compared to the other three conditions but may be misleading. Movements in the ipsilateral direction are outward when they occur in the ipsilateral field and inward when they occur in the contralateral field. Since normals are faster on inward movements it is possible that the flat function for

ipsilateral movements is due to a confound with the inward versus outward effect. In any case the data show that responses to the very same target location are faster overall when they require covert orienting in the direction ipsilateral to the lesion.

Our results suggest that each hemisphere is responsible for control of covert attention in the contralateral direction. This fits very well with the theory outlined by Kinsbourne (1977). However, with normals we have found little evidence that language tasks automatically produce a tendency to favor the right field or rightward shifts of covert attention within a field, thus not all aspects of his theory may fit our results.

The tendency of patients to show particular difficulty with reorienting toward targets contralateral to their current focus of attention does much to explain a number of conflicting results in the clinical neuropsychological literature. For example, it has long been known that right parietal patients will sometimes tend to neglect the left side of objects even when they are presented at fixation or directly to the unlesioned hemisphere. Since attention is often directed to the centroid of objects, if leftward covert scans are always difficult, one would expect to find problems with the left side of object no matter where they are presented. Since the focus of covert attention is dependent on the exact form of the object one would expect, as is observed, variability when such neglect occurs. When the gravitational straight ahead and the fixation point of the eyes are misaligned as during tests of the hemispatial neglect hypotheses (Bowers, Heilman & Van Den Abell, 1981) one would expect two contradictory

influences on the direction of covert attention , one by the fixation point and one by the gravitational straight ahead. If covert attention is not controlled by the experimenter one might expect inconsistent results that depart from a strict hemispheric solution.

A larger question is whether the covert attention system that we are studying is a module that can be engaged only by visual tasks or whether it is part of a system whose capacity is shared by different cognitive systems. If the latter one would expect engagement in non-visual tasks to influence the tendency toward poorer processing of stimuli in a direction contralateral to the lesion. This is one focus of our current patient studies.

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### Figure Captions

- Figure 1. Reaction times for six parietal patients following neutral cues (triangles) and for invalid trials with peripheral cues (circles). Filled characters are ipsilateral targets and open contralateral.
- Figure 2. Three putative mental operations involved in shifting covert attention to a target.
- Figure 3. Trial types and frequency within a block of 150 trials. Radiating lines from the hexagon indicates a cue. The star figure inside the hexagon represents a target.
- Figure 4. Reaction times as a function of target location for valid trials. Upper curves represent results of five parietal patients in fields ipsilateral and contralateral to the lesion. Lower figures are for ten normals in left and right visual field.
- Figure 5a. Reaction times for five patients in valid, cross and within field trials. The latter are broken down into within field movements in the ipsilateral and contralateral directions. Data are shown separately by visual field (e.g. ipsilateral field is on the side of the lesion and contralateral opposite the lesion). Data are from the 100 msec SOA.
- Figure 5b. Data are the same as 5a but at 600 msec SOA.

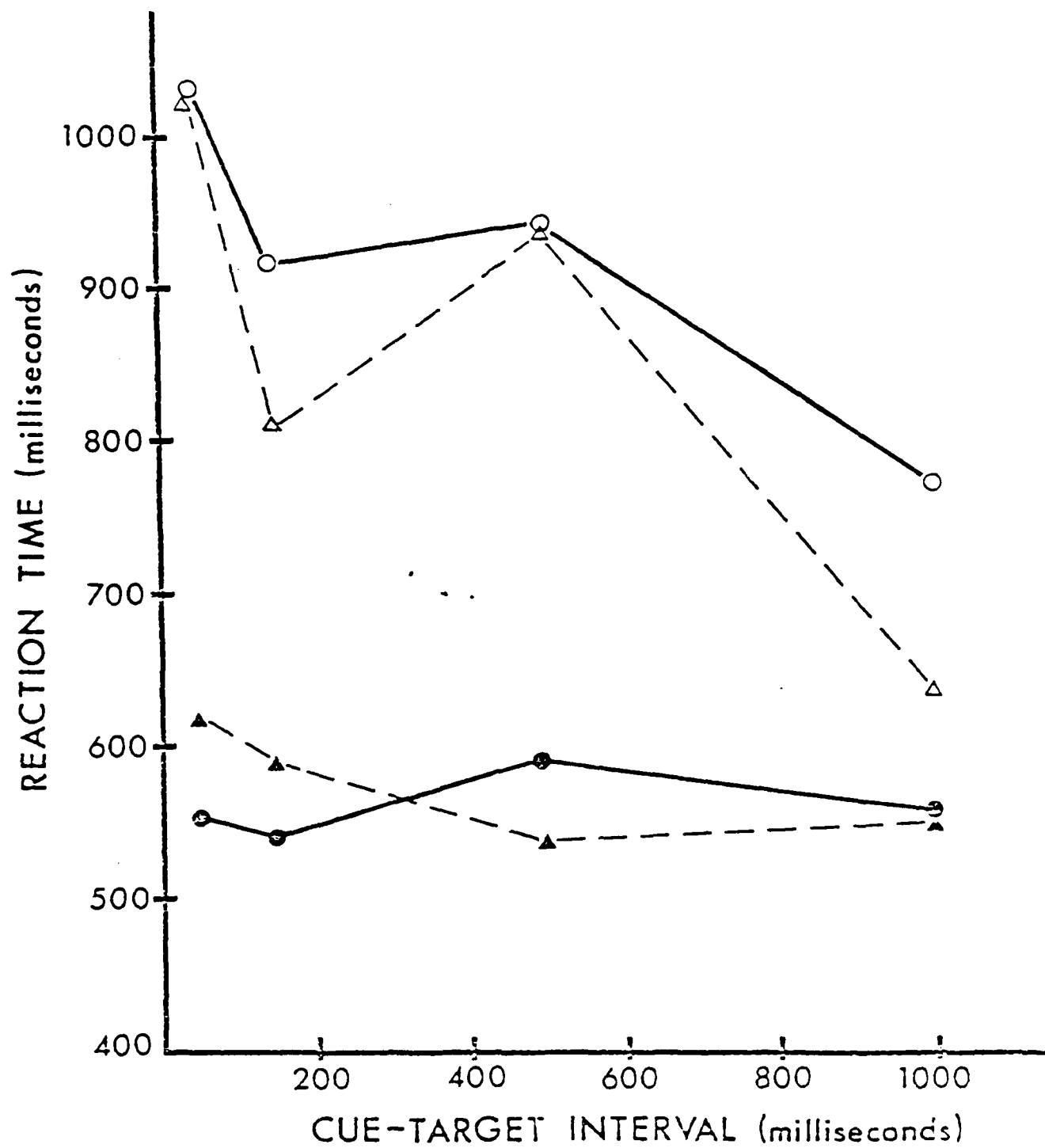


Figure 1

Operations Involved in Covert Orienting

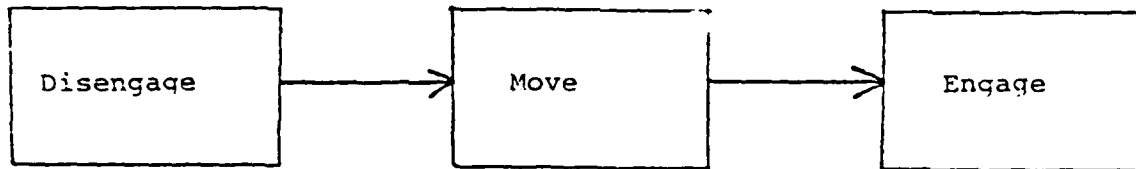
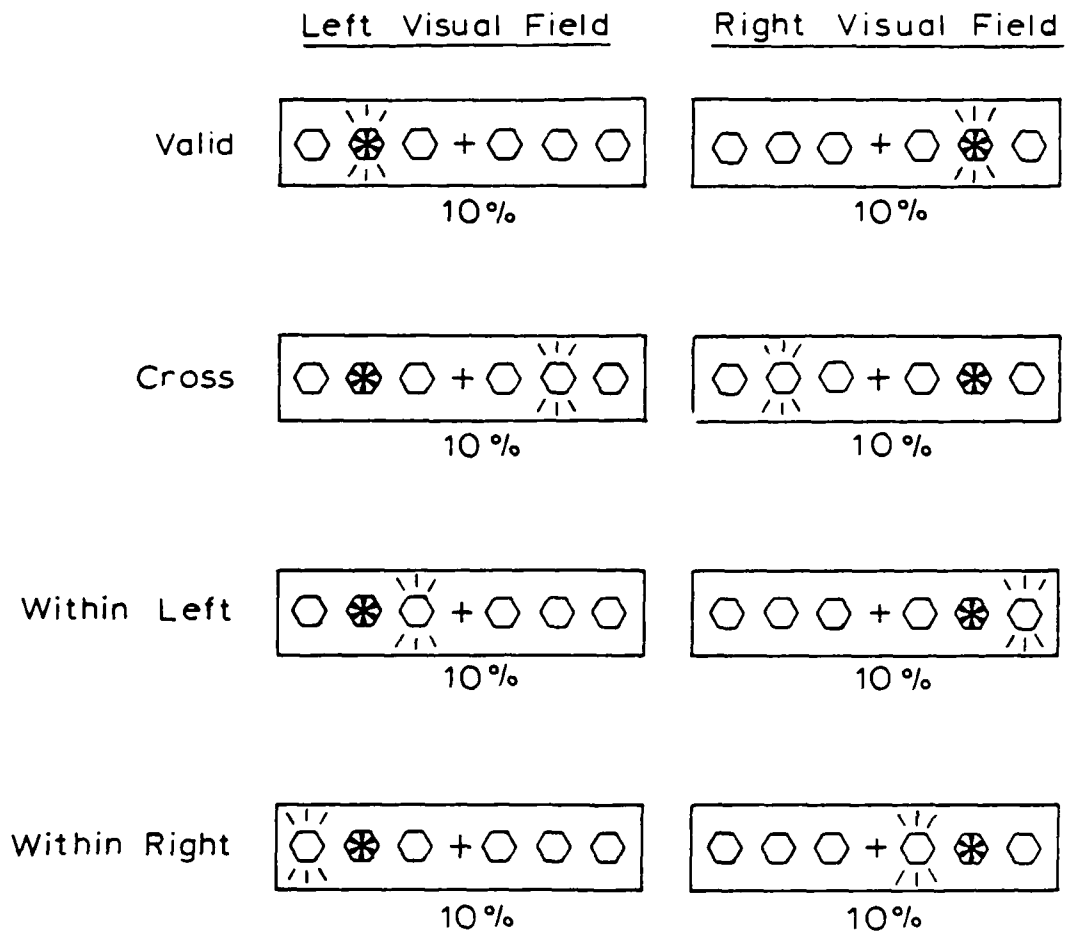


Figure 2



## CENTER TARGETS



## PERIPHERAL TARGETS

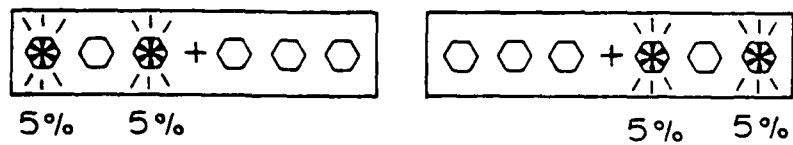


Figure 3

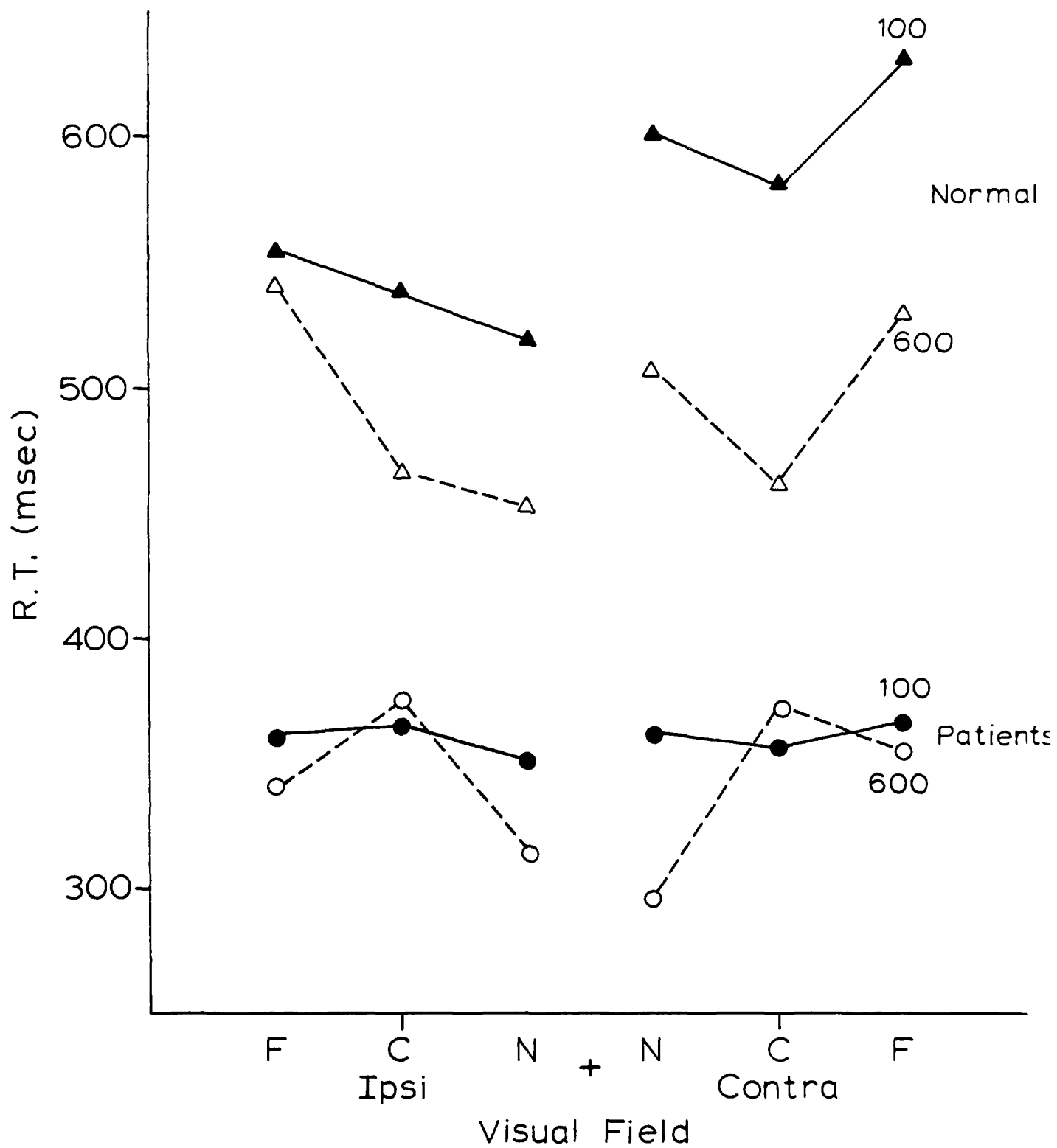


Figure 4

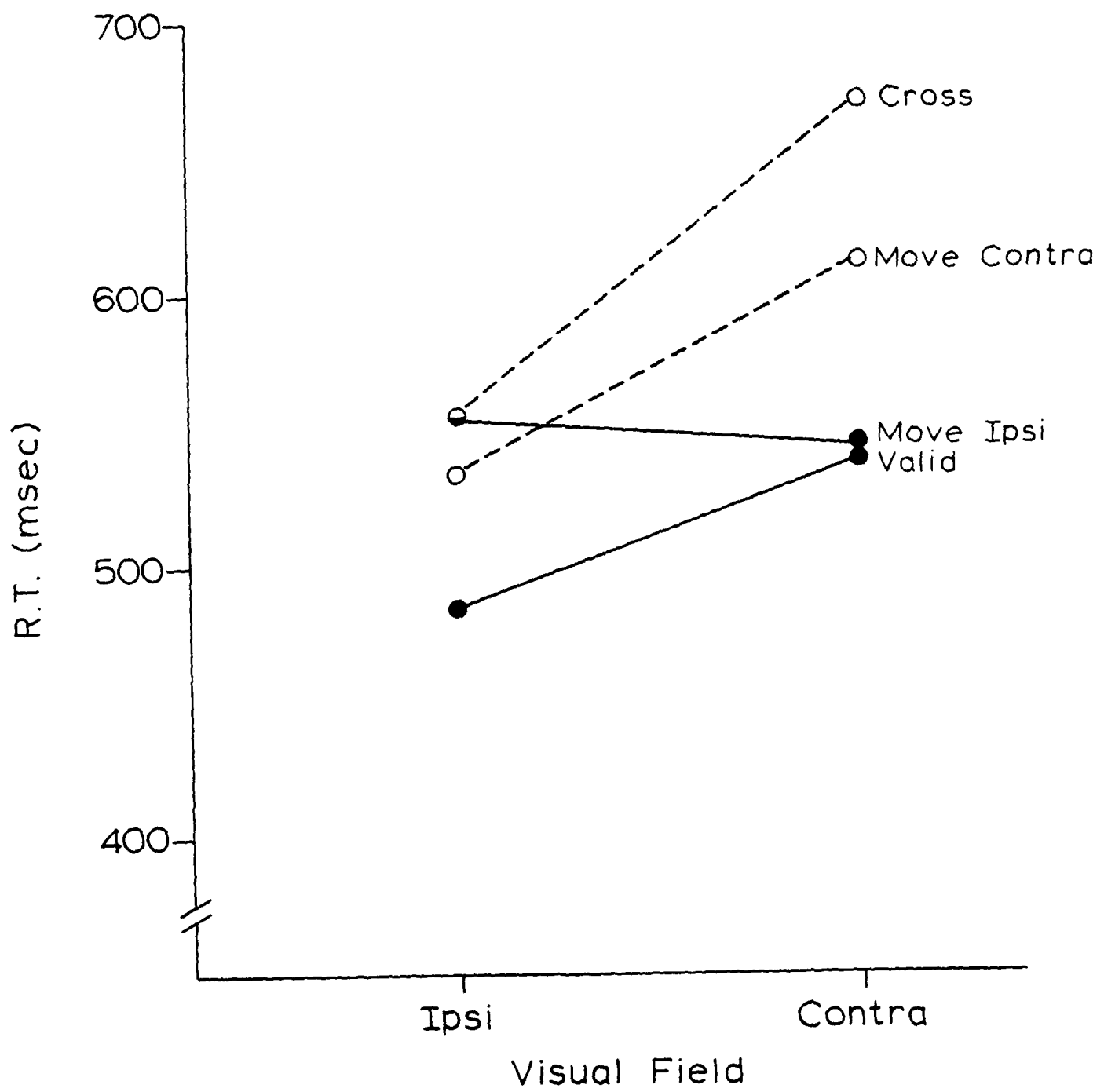


Figure 5 a

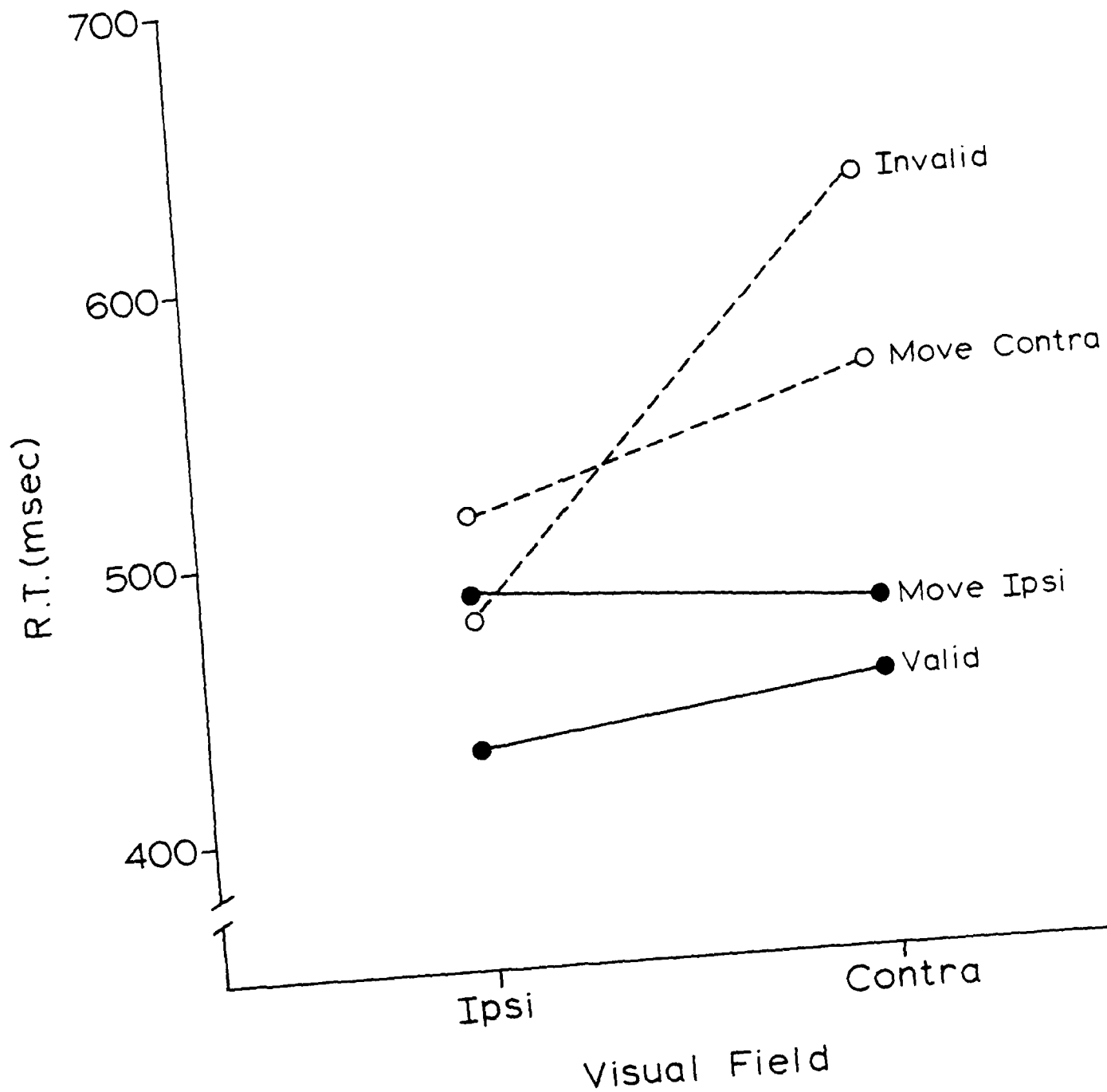


Figure 5 b

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